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Anaphylaxis due to Hymenoptera sting progressing to thoracic aortic dissection

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ABSTRACT

Here we present an 83-year-old woman with truncal pain, hypoxemia, and nausea after a Hymenoptera sting. Due to progressive truncal pain, emergent computed tomography angiography was ordered and confirmed an acute Stanford type A aortic dissection extending from the aortic root and terminating in the left common iliac artery. She was emergently transferred to a quaternary care center and managed surgically. This case highlights anaphylaxis as a unique potential trigger of aortic dissection and the need for a high index of suspicion for early diagnosis.

KEYWORDS Anaphylaxis; aortic dissection; epinephrine; Stanford type A

This case report describes an unusual presentation of anaphylaxis progressing to an acute thoracic aortic dissection. The patient met anaphylaxis clinical practice criteria, namely hypoxemia and hypotension, prompting epinephrine administration.[1] Subsequent computed tomography angiography (CTA) revealed a Stanford type A aortic dissection. Early identification of high-risk patients and definitive treatment of aortic dissection are key in reducing morbidity and mortality.

CASE DESCRIPTION

An 83-year-old woman with known hypertension, tobacco use, hyperlipidemia, and aortic regurgitation presented to emergency medical services for a Hymenoptera sting to the right hand after developing right-sided back, arm, and neck pain along with dizziness, headache, and nausea. She was noted to be hypotensive and hypoxemic on room air. The physical exam was positive for diaphoresis. She was treated with supplemental oxygen and transported.

On emergency department arrival, the patient was noted to be uncomfortable with a Glasgow Coma Scale score of 15. She was hypotensive and had an irregular heart rhythm with an aortic diastolic murmur. Her capillary refill was <2 seconds, and pulses were equal and 2+ in all extremities. The skin was diaphoretic with right-hand erythema. Her neurological exam was unremarkable. Initial laboratory results were significant for a hematocrit of 30.9%. The predominant initial concern was anaphylaxis due to persistent hypoxemia, hypotension, and nausea. Treatment was started with 0.3 mg intramuscular epinephrine, 1 L normal saline bolus, 125 mg methylprednisolone, 25 mg diphenhydramine, and 20 mg famotidine. An electrocardiogram revealed atrial fibrillation without acute ischemic changes.

Due to the discordance between worsening truncal pain and new abnormal sensation in the bilateral lower extremities, 30 minutes after arrival the patient underwent emergent CTA, which revealed an extensive Stanford type A aortic dissection *(Figure 1)*. Treatment was begun with a 10 mg intravenous labetalol push, esmolol drip of 50 mcg/kg/min, and a second normal saline 1 L bolus. Following medication administration, her blood pressure was 128/37 mm Hg; heart rate, 62 beats/min; and pulse oximetry, 100% on 2 L nasal cannula.

She was flown to a quaternary care facility without incident. On arrival she was taken directly to the operating room and underwent replacement of the ascending aorta, aortic valve resuspension, reconstruction of the proximal and distal ascending aorta, and left atrial appendage ligation. She

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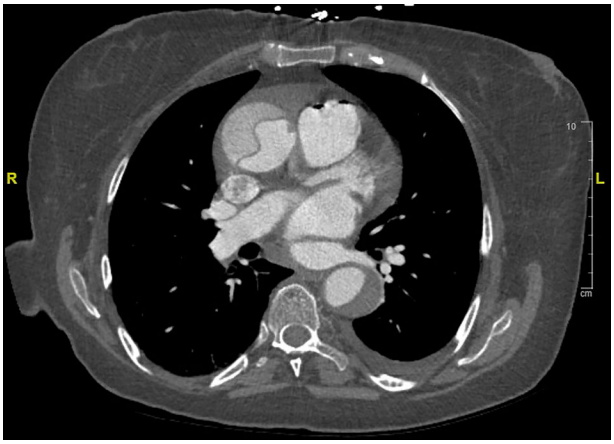

Figure 1. CTA demonstrating Stanford type A aortic dissection at level of aortic root and terminating in the left common iliac artery.

was discharged on hospital day 15 with further treatment at an inpatient rehabilitation facility.

DISCUSSION

Thoracic aortic dissections are caused by processes that promote aortic wall weakening and stress, leading to aortic wall dilation. Several risk factors in our patient are well established as driving the pathophysiology behind aortic wall weakening, such as increased age, hypertension, tobacco use, and hyperlipidemia, while aortic valve regurgitation increases aortic wall stress.[2,3]

This presentation is unique due to the combination of multiple risk factors for aortic dissection, alongside a CTA 3 months earlier that was negative for a thoracic aortic aneurysm and an initial emergency department presentation most consistent with anaphylaxis—highlighting the possibility of anaphylaxis as a novel trigger of aortic dissection. Additionally, due to the rare occurrence of aortic dissection in conjunction with anaphylaxis, this case further emphasizes the importance of understanding the possible link between anaphylaxis and aortic dissection due to the need for epinephrine use as first-line treatment in anaphylaxis.[4] The role of mast cells as a key player in anaphylaxis also suggests a possible connection with the entity of Kounis syndrome.[5]

This case presentation is the first documented instance of anaphylaxis complicated by acute thoracic aortic dissection to our knowledge. More research is needed to identify previous similar cases and delineate the significance of anaphylaxis as a trigger of acute aortic thoracic dissection.

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